56.8 per cent, while among 922 pneumonia patients without sepsis there were 131 deaths, a mortality rate of 14.2 per cent.

2. Among the causes other than sepsis responsible for death in many acute forms of the disease, cardiac-muscle failure appeared to have been a prominent factor.

3. Right ventricle dilatation was found to have occurred in 39.4 per cent of 89 lobar pneumonia autopsies and in 36.6 per cent of 112 bronchopneumonia autopsies.

4. Parenchymatous, fatty and hyaline degeneration or evidences of inflammatory reaction in the heart muscle were found in 79.3 per cent of the sections from 34 lobar pneumonia autopsies and in 59.4 per cent of the sections from 37 bronchopneumonia autopsies. The extent of these changes when considered with the right ventricle dilatation was believed in many instances to have been responsible for the circulatory failure which had occurred. In other instances the extent of the degenerative changes was less marked; in the absence of dilatation of the ventricles it appeared doubtful whether these changes, in the degree found, could have so disturbed function as to produce death.

5. The mechanical obstruction to the pulmonary circulation in extensive or massive consolidation was apparently a factor of great importance in producing right ventricle dilatation.

6. Among 213 patients without sepsis in pneumonia who were not treated by the early routine administration of a standardized tincture of digitalis the percentage of deaths believed to have been associated with cardiac failure was 25.8.

7. Among 709 patients without sepsis in pneumonia who were treated by the early routine administration of a standardized tincture of digitalis the percentage of deaths believed to have been associated with cardiac failure was 10.7.

8. The susceptibility of certain individuals to digitalis action in small dosage should be borne in mind, particularly those who manifest myocardial sclerotic changes. It should be given in large doses to elderly pneumonia patients with caution.

IMMEDIATE RECOVERY FROM EARLY DIABETES INSIPIDUS AFTER LUMBAR PUNCTURE. REPORT OF A CASE.

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Very few instances of complete recovery from diabetes insipidus after lumbar puncture have been recorded in medical literature. We are accustomed to regard the polyuria and polydipsia which so frequently accompany disturbances of the pituitary gland as symptoms of gradual, insidious onset associated with skeletal, metabolic and sexual changes. A dysfunction of this gland structure resulting in such fundamental anatomical and physiological perversions would of necessity be of long standing, and there is no doubt that most lesions of the pituitary gland are traumatic or neoplastic in origin; are more or less destructive in nature; and are accompanied by years of functional disturbance. On the other hand, suppression or alteration of pituitary function might result from the presence of an acute inflammatory process with plastic exudate or from a transient edema of the pituitary gland. Such a transient condition might account for the gradual or rapid cure of diabetes insipidus which occasionally follows the lowering of intracranial pressure by spinal puncture. Such pathological conditions of the pituitary gland may include all grades of glandular disturbance from an acute hemorrhagic infection with rapid death to a simple and transient edema. The former condition can be diagnosed correctly only at autopsy and the latter by its transient character and the early results of therapy.

The majority of investigators agree that a secretion from the pars intermedius and pars posterior of the pituitary gland in some manner or other exerts a restricting influence upon the volume output of urine. Should we accept the theory of Dr. Harvey Cushing and his associates that the pituitary secretion reaches the third cerebral ventricle by way of the infundibulum cerebri, then we can conceive that an obstruction to the flow would be produced by a local edema or by a plastic exudate and that a like obstruction would result from increased intracranial pressure due to any cause. In other words, an increased turgescence of the infundibular stalk or the back pressure from either a mild or severe hydrocephalus could act as a partial or complete obturator to the secretory flow from the pituitary gland. Even if one accepts this hypothesis, however, why a transient disturbance of pituitary function with diabetes insipidus does not commonly occur or is not more frequently recognized, remains a mystery.

Before reporting the author's case it is of interest to offer a *resume* of three cases reported in the literature, in each of which striking results followed a lowering of the intraspinal and hence of the intracranial pressure.

1. In a case reported on June 22, 1916, by Dr. Evarts Graham,[1] diabetes insipidus had existed for about two months before the patient was examined. The patient, a male, aged twenty-four years, could assign no cause for the disturbance except a blow on the chest suffered about a month and a half before the onset of polyuria. On lumbar puncture the fluid was found to be under markedly increased pressure—shooting out in a steady stream for

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[1] Jour. Am. Med. Assn., 1917, **69**, 1499.

a distance of from eight to twelve inches. Following this procedure the patient was bedridden for four days with a severe headache. At the expiration of this period he went home but returned to the hospital for five days, during which period the headache gradually disappeared. The polyuria ceased shortly after the intracranial pressure was relieved. The patient's progress after leaving the hospital was not noted.

2. A case of diabetes insipidus of four years' duration was reported by Dr. J. B. Herrick[2] in July, 1912. The patient, a male, aged forty-three years, complained of marked thirst and frequent urination. During spinal puncture the fluid escaped very slowly under low pressure, and less than 5 cc were withdrawn. This procedure was followed by great prostration, with severe headache and pain in the back of the neck, associated with anorexia, vomiting and general malaise. The pain was so intense that morphine was given. When the prostration disappeared and fluid and food were again freely taken the thirst and the output of of urine were found to be normal. Likewise, frequent attacks of petit mal which existed prior to lumbar puncture disappeared; however, Dr. Herrick was informed by the family physician three months later that these symptoms were returning.

3. A case of diabetes insipidus of about one year's duration, which had been relieved by spinal puncture, was reported recently by Dr. P. J. Cammidge.[3] The patient, a male, aged forty-two years, had been troubled with marked thirst and polyuria for fully a year before examination. About one ounce of spinal fluid, under great pressure, was removed with a needle. This event was followed by a severe headache which persisted for four days. The frequent urination rapidly disappeared, so that in a week or two the nocturnal urination had ceased. There was no recurrence of symptoms after six years.

The author's case, a male, aged twenty-seven years, was referred by Dr. R. G. Pearce, of the department of health of the B. F. Goodrich Company, Akron, Ohio. The patient came in for examination on November 26, 1920, complaining of the frequent voiding of large quantities of urine associated with great thirst. Abdominal discomfort resulting from a full bladder, had awakened him at 4 o'clock on that morning. This was immediately relieved after urination, but he was obliged to empty his bladder twice more before 6 A.M., and each time he passed a large amount of urine. Thereafter he voided copiously about every twenty minutes, even retiring frequently for relief during the physical examination.

Another symptom which persisted from the time of onset was a heavy "dry" feeling behind the bridge of the nose. As he stated it, "I feel as if trouble here is causing my thirst." He also noted

[2] Arch. Int. Med., 1912, **10**, 1–7.

[3] Practitioner, 1920, **105**, 244–247.

marked sweating of the body, hands and feet, so that a wet undershirt and soaked stockings and shoes made him feel as if he had fallen into a pool of water. Aside from these complaints the patient felt as well and active as ever, with no headache, disturbance of vision or real pain anywhere.

Past History. The patient had had typhoid fever at the age of ten years. In January, 1918, he was under observation for pulmonary tuberculosis, but was eventually discharged as negative. However, for the sake of safety, he went West for a couple of years and lived outdoors. He acquired influenza in February, 1920, but made a good recovery. Since then his health had been excellent. There was no history of venereal infection. He had been married four years. His wife and one child were living and well.

Family History. The patient's father and mother were living and well. He had four brothers and sisters, all living and in normal health. There had been no history of tuberculosis, insanity or diabetes in the family. All his grandparents lived to be over seventy-five years of age, except the paternal grandmother, who died at the age of fifty-seven years, cause of death unknown.

Physical Examination. The author's examination of the patient made four hours after the onset of the polyuria may be summarized as follows: Temperature, 97°; pulse, 70; respirations, 20; patient of medium size and build, well-developed and well-nourished, with no apparent loss in weight; mental condition alert with no undue nervousness; marked sweating of hands, body, legs, and feet, but none on the face or the neck; the shirt, stockings and shoes were soaking wet.

Head. No tenderness over the sinuses; slight ptosis of the left upper eyelid; visual fields normal to the finger test, but perimetry revealed slight concentric narrowing in the left eye; no external ophthalmoplegia; pupils equal, regular, centrally placed and reacting well to light and accommodation; vision normal by Snellen's test card; septum of nose markedly deviated to the left with absence of sense of smell on that side; slight hyperesthesia to cotton wool over the distribution of the left fifth cranial nerve; facial muscles and those of mastication negative; hearing in the left ear definitely diminished to a watch tick, with a lessened bone conduction; eardrums normal; tongue protruding in the midline; speech normal; power in sternocleidomastoids and trapezii muscles equal on both sides; teeth and tonsils negative; no evidence of increased salivation or lacrimation.

Thyroid gland palpable but not enlarged. All lymph glands of normal size.

Thorax symmetrical, expansion of upper and lower ribs as well as flaring of subcostal margins symmetrical.

Lungs negative.

Heart of normal size; no adventitious sounds; rate, 70.

Pulse rhythmic, regular, symmetrical, synchronous, with normal anacrotus and katacrotus.

Blood-pressure. Systolic, 134; diastolic, 84. Abdomen and genitalia negative.

Reflexes. Biceps, triceps, patellar and tendo Achillis equal and active; corneal, epigastric, abdominal, cremasteric and plantar reflexes all symmetrical and normal; no sensory disturbance in the skin except as noted above in the distribution of the left fifth cranial nerve; no Romberg, Babinski, Chaddock or Oppenheim; no acro- or approximo-ataxia in the upper or the lower extremities; no dysdiadokokinesia.

Urine. 2000 cc passed between 11 A.M., and 1 P.M., on the first day; clear; faint; yellowish tinge; acid; specific gravity, 1000; albumin, none; sugar, none; microscopical examination, negative.

Roentgen-ray Examination. Sella turcica normal in size and contour; skull negative for visible tumor; facial sinuses all of normal density; teeth, no caries or apical abscesses.

Eye-grounds. Left nerve-head more pale than normal, otherwise normal.

Blood Count. Red corpuscles, 5,800,000; white corpuscles, 7200; hemoglobin, 100 per cent (Tallquist).

On the second day the patient was referred to Lakeside Hospital and placed under the care of Dr. C. D. Christie. From 4 P.M., of that day to 7 A.M., of the following day the urinary output was 4100 cc, with a fluid intake of 5970 cc.

A lumbar puncture for diagnostic purposes was performed two days later, on November 30, 1920, four days after the onset of the symptoms. Eight cc of clear spinal fluid were removed under *normal pressure.* Examination of the spinal fluid showed a cell count of 2 mononuclears per cubic millimeter; globulin, negative (Noguchi, Pandy and Ross-Jones); Wassermann test on blood and spinal fluid, negative. Phenolsulphonephthalein output of kidney (after spinal puncture) amounted for the first hour to 100 cc (40 per cent), for the second hour to 500 cc (17 per cent), making the total for two hours 600 cc (57 per cent).

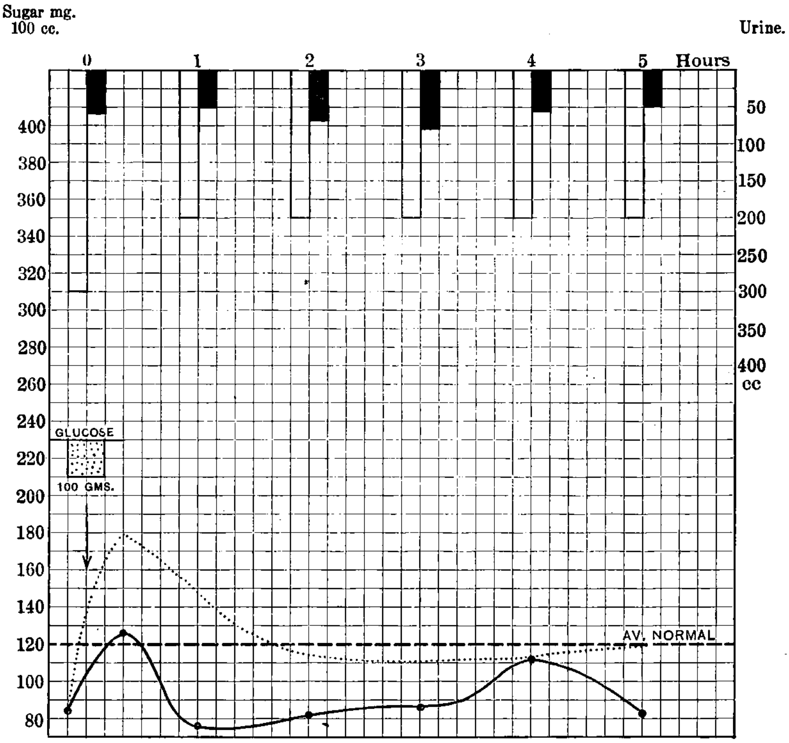
Two days later the Mosenthal nephritic test was made, showing the specific gravity of the day urine to be 1020 to 1026; of the night urine, 1032; with a total urinary output during the day of 306 cc, and during the night of 100 cc, and a liquid intake for the twenty-four hours of 1200 cc.

Thus on the second day after lumbar puncture the total urinary output for twenty-four hours was 406 cc as compared with 4100 cc during fifteen hours before the lumbar puncture; while the fluid intake during the latter period was 1200 cc as compared with 5970 cc during the first cited period. The specific gravity of the urine showed a corresponding alteration from 1005 and

1006 before the spinal puncture to 1030 immediately after, remaining at from 1026 to 1030.

The temperature was increased to 100° and 101° F. on the second and third days after the lumbar puncture, returning to normal on the fourth day.

For five days after the removal of the subarachnoid fluid the patient had a severe headache, and he was troubled with insomnia and did not feel physically fit for a period of twenty days. During this time the neurological signs gradually subsided and neither then nor since was there any recurrence of the polydipsia or polyuria. About two weeks later he developed herpes on both the upper and lower lips following an acute rhinitis. This was followed in a few days by pain under the right eye. Roentgenographs of the sinuses showed a dense clouding of the left antrum. This was tapped and a large amount of pus evacuated. Within a week



Blood-sugar curve typical of hypopituitarism shown by author's case. (Compare with normal curve shown by dotted line.) The black oblong spaces at the top indicate urinary output; the uncolored oblongs indicate water intake.

after irrigation of this sinus was begun the patient felt as well as ever and all of his neuralgic pain disappeared.

A careful examination of the patient on May 3, 1921, five and a half months after the onset of diabetes insipidus, gave normal findings throughout. He has been well and very active for over four months and has had no recurrence of his thirst or frequent urination since the spinal puncture made four days after the onset of the polyuria. The chart shows the curve of sugar tolerance at the date of this last examination. There was no hyperglycemia at any time after the ingestion of 100 gm of glucose, and at its peak the blood sugar rose only to the maximum normal level. This curve is typical of that shown by cases of hypopituitarism. However, there is no other present indication of a deficiency of pituitary secretion.

The following appear to be the significant features in the history of this patient:

1. A sudden onset of diabetes insipidus without any immediate preceding illness.
2. The subjective symptoms were a sense of discomfort behind the glabella, increased thirst, a disquieting volume in urine output and marked sweating of the body and extremities.
3. Physical examination confirmed the subjective symptoms, and, in addition, revealed involvement of the first (?), second, fifth and eighth cranial nerves on the left side.
4. Within twenty-four hours after lumbar puncture the great thirst was relieved, the urinary output was reduced to normal and the sweating ceased.
5. Examination five and a half months after the onset of the diabetes insipidus gave negative findings with the exception of laboratory evidence for hypopituitarism, revealed by the very high sugar tolerance.

What was the probable cause of the diabetes insipidus in this case? (*a*) That it was not an acute infectious process is shown by the absence of fever, leukocytosis or any other symptom prior to the spinal puncture; (*b*) that it was not a brain or pituitary tumor is shown by the absence of increased pressure of the spinal fluid and the absence of choked disk in either eye, together with the magical subsidence of symptoms after the puncture and the non-recurrence in five and a half months; (*c*) the hyperidrosis undoubtedly resulted from a transient stimulation of the bulbar-autonomic or higher centers of sweat control.

In view of these facts it seems probable that in this case the disturbance was due to a serous meningitis with edema of the infundibulum cerebri and involvement of certain cranial nerves on the left side. The removal of 8 cc of subarachnoid fluid, even though this was under normal pressure, was adequate to restore proper circulatory equilibrium. While this conclusion is of necessity entirely speculative, nevertheless the fact remains that whatever the pathological process may have been, it was sufficiently mild in character for complete relief to be secured.

Conclusions. 1. Diabetes insipidus may result not only from destructive lesions of the pituitary gland but from any cause which may obstruct the flow of the normal secretion.

2. Such an obstruction may result from a transient edema of the pituitary gland or from an inflammation with resultant plastic exudate, or it may result from extrapituitary conditions, such as increased intracranial pressure from any cause.

3. The withdrawal of spinal fluid, whether under high or low tension, tends to relieve this pressure and to remove the obstruction.

4. The experience of the author, in whose case there has been no recurrence of symptoms five and a half months after no other treatment than lumbar puncture, indicates that in any case of diabetes insipidus the patient should be given the advantage of the possibility of early relief by this means.

THE CLINICAL, BACTERIOLOGIC AND PATHOLOGIC FINDINGS IN A CASE OF INFLUENZAL MENINGITIS.

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APPARENTLY the first authentic case of influenzal meningitis was reported by Slawyk,[1] in 1899, working under the direct supervision of Pfeiffer. Occasional cases occurring thereafter are described in the literature. Writing in 1910, Simon[2] collected 12 instances of this disease.

Martha Wollstein,[3] in 1911, produced in monkeys lesions simulating influenzal meningitis. She made a review of the literature, tabulating 49 cases of pure influenzal infections. Torrey,[4] Dunn[5] [6] and Koplik[7] added 30, 4 and 6 cases respectively. Stone[8] summarized 13 others and reported a case of his own in 1920. Early in 1921, Neal[9] again reviewed the literature and discussed 32 cases, previously unreported. Since the time of her paper there have been recorded but 2 authentic instances of uncomplicated influenzal infection of the meninges, these being described by Litchfield[10] and Caprario.[11]

There have been, as far as we have been able to determine, 150